

**IN THE UNITED STATES PATENT AND TRADEMARK OFFICE**

ATTY.'S DOCKET: EISENBACH4A

In re Application of:	)	Art Unit: 1643
	)	
EISENBACH et al.	)	Examiner: L. A. Bristol
	)	
Appln. No.: 10/524,787	)	Washington, D.C.
	)	
Date Filed: September 23, 2005	)	Confirmation No. 8693
	)	
For: TUMOR ASSOCIATED ANTIGEN...	)	

**DECLARATION UNDER 37 CFR §1.132**

Honorable Commissioner for Patents  
U.S. Patent and Trademark Office  
Customer Service Window  
Randolph Building, Mail Stop  
401 Dulany Street  
Alexandria, VA 22314

Sir:

I, Lea EISENBACH, hereby declare and state as follows:

I am a co-inventor of the above-identified application and my educational and professional experience is presented in the curriculum vitae attached hereto.

I understand that there are three 35 U.S.C. §112, first paragraph, lack of enablement rejections that are maintained in the above-identified application and I will address below all three enablement issues together, as they are very closely related.

The examiner asserts that only peptides were loaded onto a single kind of APC, RMA/HHD/B7.1, and no examples of a gene-loaded APC are shown. The examiner further states that applicants' arguments rely on common knowledge for asserting art-recognized genetic engineering techniques to express peptides from APCs without any indicia of authority for this statement.

Attached hereto as an indicia of authority is the review by Lesterhuis et al., "Dendritic cell vaccines in melanoma: From promise to proof?", *Critical Reviews in Oncology/Hematology* 66:118-134 (2008). In section 7, pages 122-124, dendritic cell (DC) antigen-loading is reviewed and discussed. Although this review article was published in 2008, many of the articles cited and referred to therein were published prior to the 2003 filing date of the present application. For instance, on page 124, left column of Lesterhuis' review article, Sullenberger et al., "Emerging clinical applications of RNA", *Nature* 418:252-258 (2002), as reference [100], is cited for a novel antigen-loading technique using transfection of DC with RNA, and Nair et al., "Induction of tumor-specific cytotoxic T lymphocytes in cancer patients by autologous tumor RNA-transfected dendritic cells", *Ann. Surg.* 235:540-549 (2002), as reference [101], is cited for using tumor-derived RNA for antigen-loading. Two

additional references [106] and [107], Van Tendeloo et al., "Highly efficient gene delivery by mRNA electroporation in human hematopoietic cells: superiority to lipofection and passive pulsing of mRNA and to electroporation of plasmid cDNA for tumor antigen loading of dendritic cells", *Blood* 98:49-56 (2001) and Ponsaerts et al., "mRNA-electroporated mature dendritic cells retain transgene expression, phenotypical properties and stimulatory capacity after cryopreservation", *Leukemia* 16:13224-1330 (2002), demonstrate that RNA electroporation is highly efficient for DC transfection as a means for antigen-loading. As reported in Nair et al., "Induction of carcinoembryonic antigen (CEA)-specific cytotoxic T-lymphocyte responses *in vitro* using autologous dendritic cells loaded with CEA peptide or CEA RNA in patients with metastatic malignancies expressing CEA", *Int. J. Cancer* 82:121-124 (1999), as reference [112], equal immunogenicity was demonstrated for peptide- and RNA-pulsed DC (for antigen-loading) in colorectal cancer patients.

As positive results were obtained with the peptide-pulsed APC antigen-loading method used in Example 1 of the present specification and in the Tirosh et al. (2007) paper, previously made of record with the declaration filed September 18, 2008, one of ordinary skill in the art would conclude that the well known methods of loading APCs are interchangeable and

would fully expect the same results using any other method of antigen-loading of APCs.

The Eisenbach reference listed and attached with my previous declaration filed September 18, 2008, was presented to show that minor variants of the positively selected peptides in the present specification may occur without substantially affecting the ability to be presented by APCs and to activate CTLs.

The examiner refers to applicants wishing to obtain patent coverage for any peptide of 8-10 amino acids in length from any known or yet to be discovered TAA protein expressed by any human colon carcinoma cells formulated into any antigen presenting cell composition. However, it should be pointed out that the TAA protein must be overexpressed by human colon carcinoma cells, and the genes that are overexpressed by human colon carcinoma cells are known and available in the literature. They were known at the time the present invention was made. This is how applicants first found them. Note, for example, in the present specification on page 17, paragraph [0040] and in the first page, right column, second paragraph of the Tirosh et al. (2007) paper, referring to use of the data of Zhang et al., "Gene Expression profiles in normal and cancer cells", *Science* 276:1268-1272 (1997), a copy of which is attached hereto.

The putative TAA peptides derived from these known overexpressed genes were obtained by structural analysis, *in silico*, to find the peptides that are most likely to fit into an APC pocket. The model used was described in the present specification and in the Tirosh et al. (2007) paper. The experimental procedure of narrowing the 500 putative peptides to the ones that actually have the properties required by the claim are all described in the specification and in the Tirosh et al. (2007) paper and could be reproduced by any one of ordinary skill in the art with a lot of routine and tedious work but no undue experimentation. Nothing inventive would need to be done in reproducing the experiments. One of ordinary skill in the art reading the specification would certainly expect that if the experiment were to be repeated, one would end up with the same peptides. The experiments in Example 1 of the present specification and in the Tirosh et al. (2007) paper were conducted only with respect to HLA-A2 haplotype antigen-presenting cells and a mouse model having the human HLA-A2 gene. As the HLA-A2 haplotype is less than half of the population, based on the guidance provide by the present specification, one of ordinary skill in the art is enabled for not only the peptides specifically found in the experiments of Example 1 of the specification, which are expected to be the best peptides for the HLA-A2 population,

but also for whatever peptides would best be applicable to other APC haplotypes. This person of ordinary skill in the art would simply start from the 26 overexpressed genes and model them against the APC haplotype in question to find the putative peptides that are most likely to fit into the pocket of that APC and then repeat the tests using mice that are transgenic for that particular human haplotype of interest and using the APCs of that haplotype. This same person of ordinary skill in the art would then expect that other TAA peptides would be found and these other TAA peptides should also be part of the present invention as they can be found without undue experimentation.

The examiner states that the peptide examples are not further enabling for the "infinite genus of peptide classes encompassed by the claims." However, this statement is unfair. The starting materials are known and the 26 overexpressed proteins in human colon carcinoma cells are known. The models for finding the potentially APC-binding TAA peptides are described in the specification. The number of TAA peptides that have to be screened for any given HLA haplotype are finite and relatively small. Thus, the examiner's characterization is unjustified.

The examiner holds that the specification and the Tirosh et al. (2007) reference do not teach the amount of

experimentation required for obtaining the TAA peptides from human 1-8D interferon inducible protein 2. As shown in Table 2 in the present specification and in the Tirosh et al.(2007) paper, 24 HLA-A2.1 peptides from human 1-8D interferon inducible protein 2 were screened to identify the three TAA peptides. It should not be necessary to explain how much work was involved as the work is all described in the specification and one of ordinary skill in the art can see from the guidance provided what needs to be done. Clearly, it is a lot of work to screen the approximately 500 peptides from the 26 colorectal-associated genes indicated in Table 2 of the present specification and in Table 1 of Tirosh et al. (2007), but still, 500 is a relatively small finite number that can be screened without undue experimentation.

Nevertheless, from the guidance provided in the present specification, it is clear that the first step of finding overexpressed genes in human colon carcinoma has already been done and does not have to be repeated. The second step of finding the putative TAA peptides would have to be repeated with a different model. Models are available for some different haplotypes and this modeling is done by computer program and does not take a substantial amount of effort - a couple of weeks at most. Any university laboratory for PhD level studies in biotechnology would be equipped to do

this, and therefore this would not involve undue experimentation. The third step is for the 500, or however many there are, putative TAA peptides to be synthesized for screening. These peptides can be ordered from companies that do contract peptide synthesis, and while the cost may be expensive, obtaining such peptides is most certainly routine. The next step is to check for binding of the putative TAA peptides to APCs, which is done *in vitro* using APCs isolated from mice or by producing the APCs to the desired haplotype recombinantly. The binding tests must be done at different dilutions in 96-well plates. In general, 500 TAA peptides can be screened in 4 or 5 weeks using the standard equipment available in such a laboratory. Of course, for this routine screening, the more people working on the project, the shorter the time needed for screening.

At the same time or following the screening of the putative TAA peptides, CTLs that can be used in the following step are produced by immunizing a mouse, humanized with the HLA haplotype being tested, with human carcinoma extract and isolating the CTLs from the spleen. Then each of the peptides that were found positive in the previous screen are loaded on the APC of the desired haplotype and incubated together with the CTLs to see if the APCs die. Although this represents a lot of tedious work and although it also may take six months



or more for a single PhD student to complete the testing, one of ordinary skill in the art would clearly recognize that it is something that a PhD student could be taught to do and that it could be done without undue experimentation. Again, the more people working on the project, the shorter the time involved. No inventive steps are necessary to complete such a screen. This screening narrows the 500 putative TAA peptides down to probably 20-30 different TAA peptides. The next step is to take the TAA peptides that were positive in the previous step, load them onto APCs and actually immunize the mice with the peptide-loaded APCs. One would again isolate the CTLs and test for specificity against human colon carcinoma cells. The amount of time depends on how many people are working on it and how many animals are being used simultaneously. It took my laboratory one year to do this but it could have been done faster with more people and more funding. The work was certainly not "undue," but rather involved standard routine experimental work. In the experiments reported in Example 1 of the present specification, we ended up with seven TAA peptides that were positive, all seven of which fall within the scope of the present claims. For the further experiments, my laboratory happened to decide to concentrate on three of the identified TAA peptides.

In short, while there may be a substantial amount of work involved in repeating the process described above (with guidance provided in the present specification) on another haplotype, it would not take undue experimentation to do so and to find TAA peptides which would be usable in that portion of the population.

The undersigned declares further that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

6/16/2009  
Date

/Lea Eisenbach/  
Lea EISENBACH

## **LEA EISENBACH, PhD**

### **CURRICULUM VITAE**

#### **EDUCATION**

- 1962-66      Technical High School of Engineering, Tel Aviv University, Tel Aviv, Israel. Subject of thesis: Immunospecific antibodies to nucleosides.
- 1966              Awarded degree in Practical Chemical Engineering.
- 1966-69       Undergraduate studies in the Department of Chemistry, Tel Aviv University.
- 1969              Awarded B.Sc. degree, Tel Aviv University.
- 1969-71       Graduate studies towards M.Sc. degree, Department of Biochemistry, Tel Aviv University. Subject of thesis: "The Modification of Porcine Carboxypeptidase B by P-azo-benzenearsonate." Supervisor: Prof. M. Sokolovsky.
- 1972              Awarded M.Sc. degree, Tel Aviv University.
- 1973-78       Graduate studies towards Ph.D. degree at the Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel. Subject of thesis: "On the Mechanism of Glucose-6-Phosphate Dehydrogenase Regulation in Mouse Liver." Supervisor: Prof. G. Yagil.
- 1979              Awarded Ph.D. degree, Feinberg Graduate School of the Weizmann Institute of Science.
- 1978-79       Postdoctoral training, The Institute for Enzyme Research, Madison, Wisconsin, USA. Research associate with Prof. M. Nomura. Research interests: Effects and mechanisms of ppGpp on transcription of E. coli ribosomal RNA operons in an in vitro system.
- 1979-80       Postdoctoral training, Department of Biochemistry, University of Wisconsin, Madison, Wisconsin, USA Position: Research Associate with Profs. D.L. Nelson and C. Kung. Research interests: The biochemistry of behavior of Paramecium tetraurelia. Mapping of proteins in the ciliary membrane which are functional in regulation of swimming.

## **POSITIONS**

- 1980-83      Research Associate with Professor Michael Feldman, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel. Research interest: Tumor metastases.
- 1983-86      Staff Assistant Scientist with Professor Michael Feldman, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.
- 1986-1989    Staff Associate Scientist, with Professor Michael Feldman, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.
- 1989-1994    Staff Senior Scientist, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.
- 1994-1995    Associate Professor, Dept. of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.
- 1995          Associate Professor, Dept. of Immunology, The Weizmann Institute of Science, Rehovot, Israel (rearrangement of staff in departments).

## **MISCELLANEOUS**

- 1966-67      Army service.
- 1970-72      Non-degree studies in the Department of Education, Tel Aviv University.
- 1972          Teaching certificate in Chemistry.
- 1969-70      Teaching, chemistry, physics, high school level.
- 1969-72      Teaching, mathematics at "Seminar Hakibbutzim", Tel Aviv (College level).
- 1969-71      Teaching assistant, Department of Biochemistry, Tel Aviv University.
- 1971-73      Research assistant in a joint program for the Department of Chemical Immunology of the Weizmann Institute and the Immunology Laboratory at the Beilinson Hospital, Petah-Tikva, Israel.
- 1983          Mini-sabbatical, Dr. Robert Gallo, NCI, Bethesda, Maryland. Subject: Oncogene Expression in Metastases.
- 1988          Mini-sabbatical, Dr. Eli Gilboa, Memorial Sloan Kettering, New York, Subject: Gene transfer by retroviral vectors.

- 1995-1998 Secretary general of the Israeli Immunological Society.
- 1997 Review Board Free University, Brussels, Belgium.
- 1998 Review committee ACSBI, YY, TCRF-UICC.
- 1998-2001 President of the Israel Immunological Society.

## **PROFESSIONAL SOCIETY MEMBERSHIPS**

Israel Immunological Society.  
Secretary general (1995-1998, President 1998 -2001).

International Metastasis Research Society.

American Association for Cancer Research.

European society for gene therapy.  
Chairperson of the committee of gene therapy and immunotherapy of cancer (2000-2003).

UICC.  
(Reviewer Board 98-03).

## **EDITORIAL BOARDS**

Gene Therapy.  
Karol Sikora, Bob Williamson, Joseph Glorioso (Eds). Macmillan Magazines Ltd.

Clinical and experimental metastasis.  
Suzanne A Eccles, Garth L Nicolson, Tatsuro Irimura (Eds), Kluwer Pub.

## **REVIEWER (Journals)**

PNAS  
Journal of Immunology  
Oncogene  
International Journal of Cancer  
Clinical and Experimental Metastasis  
British Journal of Cancer  
International Immunology  
Gene Therapy  
Cancer gene therapy  
Human gene therapy  
European J Immunology  
European J Cancer

Immunology letters  
FEBS journal  
Cancer letters  
Molecular Medicine

### **INVITED PRESENTATIONS (2003-2008)**

Keystone Symposia, Tumor Immunology (C5) February, 17-23, 2003, Keystone, USA.

The 34th symposium of princess Takamatsu cancer research fund, November 11-15, 2003, Tokyo, Japan.

Vaccines, present and future. November 27-29, 2003, Dead Sea resort, Israel

CapCure Israel Scientific Retreat, January 13-16, 2004, Ein Gedi, Israel.

The 33rd annual meeting of the Israel Immunological Society, February 25-26, 2004, Bar-Ilan University, Israel.

The Seventh International Conference of Anticancer Research, October 25-30, 2004, Corfu, Greece.

Israel Vaccine Research Initiative (IVRI), June 21, 2005, Jerusalem, Israel.

Immune Mediated diseases: from Theory to therapy. October 3-8, 2005, Moscow, Russia.

Cancer, Metastasis, AIDS and Immunotherapy, ISF workshop in memory of Professor Michael Feldman. March 13-14, 2006. Rehovot Israel. (Meeting chairperson).

Tumor Immunology, May 17-21, 2006. Halle, Germany.

The scientific legacy of Shraga Segal, a symposium to his memory, February 21, 2007, Beer-Sheba, Israel.

The 14<sup>th</sup> international conference of cryosurgery, the 1<sup>st</sup> international conference on cryoimmunology, November 3-6, 2007. Beijing, China.

Cancer-a cellular and molecular view, M.D. Moross Cancer Institute of the WIS and the McGill Cancer Centre, McGill University, Montreal, October 26-27, 2008. Rehovot, Israel.

### **GRANTS AND AWARDS (2002-2008)**

ICA (1/2001-12/2002)

MHC class I and class II restricted antigens in immunotherapy of cancer.

Ornest Family Foundation (3/2001-2/2002)  
Carcinoma TAAs-biology and immunotherapy.

Sterenber Fund (8/2001-7/2002)  
1-8D gene and colon carcinoma.

ISF(10/2001-9/2005)  
The role of interferon inducible gene 1-8D in tumorigenicity and immunogenicity of colon cancer.

Minerva (1/2002-12/2004)  
The role of interferon-inducible 1-8D gene in tumorigenicity and immunogenicity of colon cancer.

Levin Fund (2/2002-12/2002)  
Glycopeptides as targets for immunotherapy of breast and ovarian carcinomas.

Lombroso Grant for Clinical Cancer Research (4/2002-3/2003)  
Novel colon carcinoma TAAs-biology and immunotherapy.

Moross Institute for Cancer Research (4/2002-3/2003)  
Novel colon carcinoma TAAs-biology and immunotherapy

Ministry of industry and commerce (Nofar) (8/2002-7/2003)  
Anti colon carcinomas vaccines based on novel human tumor associated antigen 1-8D peptides.

Ornest Family Foundation (9/2002-8/2003)  
Novel anti prostate cancer vaccines based on tumor associated antigen peptides.

ICRF (9/2002-8/2004)  
The role of 1-8 family of interferon inducible gene in carcinogenesis and immunotherapy.

ICA (1/2003-12/2004)  
Peptides and glycopeptides as targets for anti cancer immunotherapeutic approaches in breast and ovarian carcinomas.

Weizmann Institute-Excellence Center (10/2003-9/2004)  
Characterization of new human carcinoma antigens (TAAs) and of respective T cell memory and effector cell responses.

Horowitz (1/2004-2/2005)  
Novel anti prostate cancer vaccines based on tumor associated antigen peptides.

Lewis Family Charitable Trust (3/2004-2/2008)  
Anti colon-carcinoma vaccines based on tumor associated antigens (TAA) peptides.

Horowitz (1/2005-12/2005)  
The role of normal and mutant 1-8D interferon inducible gene in carcinogenesis and immunotherapy.

ICRF (9/2005-8/2007)  
The role of the 1-8 interferon inducible genes in tumor progression.

ISF (10/2005-9/2009)  
Small interferon stimulated genes: Role in cancer and immunity.

Ministry of health (3/2007-2/2009)  
The effect of SNPs in tumor-associated antigens on the immunogenicity of peptide based vaccines.

Moross Institute for Cancer Research (7/2007-6/2008)  
T-cell receptor evolution for tumor immunotherapy.

Yeda research and development (7/2007-6/2008)  
Preparation of glioma immunotherapy clinical trial-glioma separation and characterization, and immune monitoring of patients.

Lewis Family Charitable Trust (3/2008-2/2009)  
Anti colon-carcinoma vaccines based on tumor associated antigens (TAA) peptides.



## **STUDENTS**

### **Ph.D. degree**

Gelber Cohava (graduated 1989)  
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Katz Anne (graduated 1994)  
Plaksin Daniel (graduated 1994)  
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Simons Arnon (in collaboration with Prof. V. Rotter, graduated 1998)  
Feigelson Sara (in collaboration with Prof. Z. Eshhar, graduated 1999)  
El-Shami Khaled (graduated 2000)  
Carmon Lior (graduated 2001)  
Tirosh Boaz (graduated 2002)  
Bar-Haim Erez (graduated 2003)  
Lee Sung-Hyung (graduated 2005)  
Machlankin Arthur (graduated 2005)  
Farago Marganit (left 2003)  
Volovitz Ilan (in collaboration with Prof. I. Cohen, graduated 2007)  
Goldberger Ofir (graduated 2008)  
Daniel Vered (graduated 2008)  
Avraham Efrat  
Cohen Noam (in collaboration with Dr Steffen Jung)  
Boxbaum Yonathan  
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### **Post-docs**

Stepensky David (2003-2005)  
Avrahami Dorit (2004-present)  
Daniel-Carmi Vered (2008-present)

### **M.Sc. degree**

Plaksin Daniel (graduated 1987)  
Gubbay John (graduated 1988)  
Porgador Angel (graduated 1989)  
Mandelboim Ofer (graduated 1991)  
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Erel Naomit (graduated 1993)  
Feigelson Sara (graduated 1993)  
Kopcow Hernan (graduated 1996)  
Morag Tamar (in collaboration with Prof. M. Fridkin, graduated 1996)  
Bar-Haim Erez (graduated 1997)  
Popovich Dan (graduated 1998)

Shachar Hadas (graduated 1998)  
Stabinsky Michal (graduated 1998)  
Volovitz Ilan (graduated 2000)  
Priel Irene (graduated 2001)  
Goldberger ofir (graduated 2002)  
Avraham Efrat (graduated 2003)  
Daniel Vered (graduated 2003)  
Finkel Eran (graduated 2003)  
Windrech Ruty (graduated 2004)  
Guttman Chen (graduated 2005)  
Sheiket Helena (graduated 2005)  
Tavori Hagai (graduated 2007)  
Karp Noga (graduated 2008)  
Reuven Eliran  
Azulay Meir  
Bordigary Giovanna  
Vidiborsky Zoya

## **LEA EISENBACH, PhD**

### **PUBLICATIONS 72-08**

1. Sokolovsky, M., Eisenbach, L. (1972). Porcine carboxypeptidase B Arsanilzocarboxypeptidase, spectral and functional consequences of modification of tyrosine-248. *Eur J Biochem* 25:483-490.
2. Eisenbach, L., Yagil, G. (1975). Purification of glucose-6-phosphate dehydrogenase from induced and repressed mouse liver. *Isr J Med Sci* 11:1175.
3. Eisenbach, L., Shimron, F., Yagil, G. (1976). The effect of age on the regulation of glucose-6-phosphate dehydrogenase in mouse liver. *Exp Gerontol.* 11:63-71
4. Eisenbach, L., Eisenbach, M. (1979). Electrophoretic mobility of membrane fragments on a sucrose gradient. Application to isolated purple membrane fragments from *Halobacterium halobium*. *Anal Biochem* 92:228-232.
5. Eisenbach, L., De Baetselier, P., Katzav, S., Segal, S., Feldman, M. (1983). Immunogenic control of metastatic competence of cloned tumor cell populations. *Symp Fundam. Cancer Res.* 36:101-121.
6. Eisenbach, L., Ramanathan, R., Nelson, DL. (1983). Biochemical studies of the excitable membrane of *Paramecium tetraurelia*. IX. Antibodies against ciliary membrane proteins. *J Cell Biol* 97:1412-1420.
7. Eisenbach, L., Segal, S., Feldman, M. (1983). MHC imbalance and metastatic spread in Lewis Lung carcinoma clones. *Int J Cancer* 32:113-120.
8. Eisenbach, L., Hollander, N., Greenfeld, L., Yakor, H., Segal, S., Feldman, M. (1984). The differential expression of H-2K versus H-2D antigens, distinguishing low metastatic from high metastatic clones, is correlated with the immunogenic properties of the tumor cells. *Int J Cancer* 34:567-5731.
9. Eisenbach, L., De Baetselier, P., Katzav, S., Segal, S., Feldman, M. (1984). Immunogenetic control of metastatic competence of cloned tumor cell populations. In: "Cancer Invasion and metastasis: Biologic and Therapeutic Aspects," eds Nicolson GL, Milas L, Raven Press, New York, pp 101-121.
10. Eisenbach, L., Segal, S., Feldman, M. (1985). Proteolytic enzymes in tumor metastasis. I. Plasminogen activator in clones of Lewis lung carcinoma and T10 sarcoma. *J. Natl. Cancer Inst.* 74:77-86.

11. Eisenbach, L., Segal, S., Feldman, M. (1985). Proteolytic enzymes in tumor metastasis. II. Collagenase type IV activity in subcellular fractions of cloned tumor populations. *J. Natl. Cancer Inst.* 74:87-93.
12. Eisenbach, L., Hollander, N., Segal, S., Feldman, M. (1985). The differential expression of class I MHC antigens controls the metastatic properties of tumor cells. *Transpl. Proc.* 17:729-734.
13. Eisenbach, L., Feldman, M. (1985). Genes and antigens controlling tumor metastasis. In: *Haematology and Blood Transfusion, Modern Trends in Leukemia, VI* (Neth R, Gallo R, Greaves M, Jenkins A, eds). Vol 29, Springer Verlag, Berlin, pp. 449-507.
14. Eisenbach, L., Feldman, M. (1986). Enzymes, receptors, oncogenes in high- and low-metastatic tumor clones. In: *New Experimental Modalities in the Control of Neoplasia* (Chandra P, ed.) Plenum Press, New York, pp 57-70.
15. Eisenbach, L., Katzav, S., Feldman, M. (1986). Immunomodulation of tumor metastasis. *Ibidem*, pp 81-90.
16. Dvorat, A., Scharf, J., Eisenbach, L., Gershon, D. (1986). G6PD molecules devoid of catalytic activity are present in the nucleus of the rat lens. *Exp. Eye Res.* 42:489-496.
17. Eisenbach, L., Kushtai, G., Plaksin, D., Feldman, M. (1986). MHC genes and oncogenes control the metastatic phenotype of tumor cells. *Cancer Rev.* 5:1-8.
18. Barzilay, J., Kushtai, G., Plaksin, D., Feldman, M., Eisenbach, L. (1987). Expression of major histocompatibility class I genes in differentiating leukemic cells is temporally related to activation of c-fos protooncogene. *Leukemia* 1:198-204.
19. Feldman, M., Eisenbach, L. (1987). Molecular controls of tumor metastasis. In: *Accomplishments in Cancer Research 1986* (Fortner JG, Rhoads JE, eds.) JR Lippincott Co., Philadelphia, pp 194-200.
20. Kushtai, G., Barzilay, J., Feldman, M., Eisenbach, L. (1988). The c-fos proto-oncogene in murine 3LL carcinoma clones controls the expression of MHC genes. *Oncogene* 2:119-128.
21. Plaksin, D., Gelber, C., Feldman, M., Eisenbach, L. (1988). Reversal of the metastatic phenotype in Lewis lung carcinoma cells following transfection with syngeneic H-2K<sup>b</sup> gene. *Proc. Natl. Acad. Sci. USA* 85:4463-4467.
22. Feldman, M., Eisenbach, L. (1988). Genes controlling the metastatic phenotype. *Cancer Sur.* 7:555-572.

23. Eisenbach, L., Gubbay, J., Gelber, K., Kushtai, G., Feldman, M. (1988). Do oncogenes play a role in tumor metastasis? In: Cancer Metastasis: Biological and Biochemical Mechanisms and Clinical Aspects (Hellman K, Liotta LA, Prodi G, eds.) Plenum Publ. Corp. New York, pp 281-291.
24. Feldman, M., Plaksin, D., Gelber, C., Kushtai, G., Eisenbach, L. (1988). Control of MHC genes that regulate the metastatic phenotype of tumor cells. In: Cancer Metastasis: Biological and Biochemical Mechanisms and Clinical Aspects (Hellman K, Liotta LA, Prodi G, eds). Plenum Publ. Corp., New York, pp 281-291.
25. Feldman, M., Gelber, C., Plaksin, D., Kushtai, G., Eisenbach, L. (1988). The reversal of the metastatic phenotype by gene transfer. Proc. CIBA Found. Symp. on Metastasis, London pp 170-189.
26. Feldman, M., Eisenbach, L. (1988). The metastatic phenotype of cancer cells. Sci. Am. Vol. 256 No. 11:60-85.
27. Feldman, M., Plaksin, D., Gelber, C., Kushtai, G., Eisenbach, L. (1988). Control of MHC genes that regulate the metastatic phenotype of tumor cells. Adv Exp Med Biol 233:109-117.
28. Gelber, C., Plaksin, D., Vadai, E., Feldman, M., Eisenbach, L. (1989). The abolishment of metastasis formation by tumor cells transfected with "foreign" H-2K genes. Cancer Res. 49:2366-2372.
29. Tzehoval, E., Dagan, S., Eisenbach, L., Atsmon, J., Feldman, M. (1989). Immunogenic properties of macrophage hybridomas. Eur J Immunol 19:89-96.
30. Martinez, RD., Eisenbach, L., Feldman, M. (1989). Cytotoxic and proliferative effect of tobacco products on Lewis lung adenocarcinoma cells and spleen lymphocytes. Allergol Immunopathol 17:257-261.
31. Porgador, A., Feldman, M., Eisenbach, L. (1990). H-2K<sup>b</sup> transfection of B16 melanoma cells result in reduced tumorigenicity and metastatic competence. J. Immunogenet 16:291-303.
32. Kushtai, G., Feldman, M., Eisenbach, L. (1990). c-fos transfection of 3LL tumor cells turns on MHC gene expression and consequently reduces their metastatic competence. Int. J. Cancer 45:1131-1136.
33. Feldman, M., Eisenbach, L. (1990). Metastases unveiled (in Hebrew). Mada Vol. 34, No. 4, pp. 174-179.
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